

Author's response to reviews

Title: Acute pancreatitis following medical abortion: Case report

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PDF covering letter

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Dear Editor,

Thank you for your interest in and critique on our manuscript "Acute pancreatitis following medical abortion: Case report" with reference number 9762123462710955. We have tried to reply to the critique raised by your Referees. Please find our revised manuscript uploaded to your server.

Our reply will be as follows:

To Dr Hobart Harris:

Minor essential revisions:

- 1) Fig 1 is now omitted, and the essential information is instead included in the text, as suggested. Line 5 and 6 of paragraph 3 on page 3 now contains this information and thus differs somewhat from the original manuscript.
- 2) The limitations of a CT scan for imaging gallstones are now commented on on line 11 of paragraph 3 on page 3.
- 3) We do not have lipase levels.

Major compulsory revisions:

- 1) The patient's pancreatitis was indeed life-threatening, and demanded 14 days of intensive care to control. It was classified as severe acute necrotizing pancreatitis. However, classification according to Ransons, APACHE II, Glasgow or Balthazar was not made. We have tried to be clearer on just how severe the patient's condition was on line 8-10, 12-15 and 19 of paragraph 3 on page 3.

To Dr Paul Georg Georg Lankisch:

The reference interval of plasma amylase at our hospital is 0.2-0.8 $\mu\text{kat/L}$, which is now given on line 10 of paragraph 3 on page 3.

Finally, we have also tried to improve the language throughout the manuscript, and corrected a flaw, i.e. that the patient's highest measured p-amylase level was 23 $\mu\text{kat/L}$, as it says in table 1. We hope that we have adequately responded to the critique raised, and that the manuscript now is suitable for publication in *BMC Women's Health*.

Sincerely yours,

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Editor:

(1): Due to the positive finding concerning the almost two-fold difference between genotypes in mean LVMI change, we reason that the inclusion of data from power calculations are not necessary, since a power calculation usually is only interesting for the evaluation of a possible type II bias in small studies showing no difference between categories. Our aim has been to identify polymorphisms that correlate to clinically significant, large differences between genotypes in antihypertensive drug response, as was the case, for instance, in the article "Polymorphisms in the angiotensinogen and angiotensin II type 1 receptor gene are related to change in left ventricular mass during antihypertensive treatment" by Kurland et al in a recent number of Journal of Hypertension. In that article, Kurland et al report a marked difference in LVMI change between genotypes. Since the change in LVMI is the main point in this manuscript and the difference between genotypes was almost 100%, we have not included data from power calculations. However, we present them in this letter for you to evaluate. This study thus had an 80% power to predict a little less than 20% difference in LVMI change at 48 weeks within 95% CI.

	Difference (%)	n in each group
LVMI change at 48 weeks	20	11
	15	20
	10	44

(2): In one of the subgroups, the Arg/Arg genotype in the Irbesartan treatment group, baseline LVMI was not completely normally distributed when normality was analyzed by the Shapiro-Wilks test. This, however, did not affect our conclusions. There was no significant baseline difference in LVMI between the Arg/Arg and Arg/Lys group given Irbesartan. The p value for a difference between the groups with a t-test was 0.32 for the original data and 0.43 for the log-transformed data. The p values for the crude and the multivariate adjusted difference in mean LVMI difference after 48 weeks between the groups remained nearly identical after log transformation of the LVMI data, and therefore skewness can not explain our finding. The LVMI data in the other subgroups displayed a normal distribution by the original data. The second paragraph on page 5 in the Discussion part has been added, explaining this.

(3): The adjusted mean values were actually calculated by regression analysis, i.e. procedure GLM of the SAS software. This method uses multivariate linear regression analysis to calculate the adjusted mean values of the dependent variable.

(4): Yes. Please refer to the following articles:

a: Hallberg P, Lind L, Michaelsson K, Karlsson J, Kurland L, Kahan T, Malmqvist K, Ohman KP, Nystrom F, Melhus H. *B2 bradykinin receptor (B2BKR) polymorphism and change in left ventricular mass in response to antihypertensive treatment: results from the Swedish Irbesartan Left Ventricular Hypertrophy Investigation versus Atenolol (SILVHIA) trial.* J Hypertens. 2003 Mar;21(3):621-4.

b: Hallberg P, Karlsson J, Kurland L, Lind L, Kahan T, Malmqvist K, Ohman KP, Nystrom F, Melhus H. *The CYP2C9 genotype predicts the blood pressure response to irbesartan: results from the Swedish Irbesartan Left Ventricular Hypertrophy Investigation vs Atenolol (SILVHIA) trial.* J Hypertens. 2002 Oct;20(10):2089-93.

c: Kurland L, Melhus H, Karlsson J, Kahan T, Malmqvist K, Ohman P, Nystrom F, Hagg A, Lind L. *Aldosterone synthase (CYP11B2) -344 C/T polymorphism is related to antihypertensive response: result from the Swedish Irbesartan Left Ventricular Hypertrophy Investigation versus Atenolol (SILVHIA) trial.* Am J Hypertens. 2002 May;15(5):389-93.

d: Kurland L, Melhus H, Karlsson J, Kahan T, Malmqvist K, Ohman P, Nystrom F, Hagg A, Lind L. *Polymorphisms in the angiotensinogen and angiotensin II type 1 receptor gene are related to change in left ventricular mass during antihypertensive treatment: results from the Swedish Irbesartan Left Ventricular Hypertrophy Investigation versus Atenolol (SILVHIA) trial.* J Hypertens. 2002 Apr;20(4):657-63.

e: Kurland L, Melhus H, Karlsson J, Kahan T, Malmqvist K, Ohman KP, Nystrom F, Hagg A, Lind L. *Angiotensin converting enzyme gene polymorphism predicts blood pressure response to angiotensin II receptor type 1 antagonist treatment in hypertensive patients.* J Hypertens. 2001 Oct;19(10):1783-7.

Reviewer:

We hope that the association between ALAP and angiotensin II blockade is now clearer after the expansion of sentence 2 in the third paragraph in the Discussion part on page 5. We have also added sentence 2 in the fourth paragraph in the Discussion part on page 6, where the possibility of linkage disequilibrium is mentioned. When the small lys/lys group is combined with the lys/arg group the difference between carriers of the lys allele and the arg/arg group becomes as follows:

Univariate	<i>LVMI change at 48 weeks</i>	<i>SE</i>	<i>p</i>
Lys/Lys & Lys/Arg	-14.5	6.0	0.02
Arg/Arg	-33.6	5.4	

Multivariate	<i>LVMI change at 48 weeks</i>	<i>SE</i>	<i>p</i>
Lys/Lys & Lys/Arg	-18.9	4.1	0.06
Arg/Arg	-30.2	3.6	

Thus, the combination has minor effect on the results.

The abbreviations mentioned have now been expanded.